PATHOPHYSIOLOGY OF RENAL COLIC

Renal colic affects approximately 1.2 million people each year and accounts for approximately 1% of all hospital admissions. The overall lifetime rate of kidney stones in the general population is approximately 12% for men and 4% for women. Having a family member with a history of stones doubles these rates.

The colicky-type pain known as renal colic usually begins in the upper lateral mid back over the costovertebral angle and occasionally subcostally. It radiates inferiorly and anteriorly toward the groin. The pain generated by renal colic is primarily caused by the dilation, stretching, and spasm caused by the acute ureteral obstruction. (When a severe but chronic obstruction develops, as in some types of cancer, it is usually painless.)

Colic is a misnomer because renal colic pain tends to remain constant, whereas intestinal or biliary colic is usually somewhat intermittent and often comes in waves. The pattern of the pain depends on the individual's pain threshold and perception and on the speed and degree of the changes in hydrostatic pressure within the proximal ureter and renal pelvis. Ureteral peristalsis, stone migration, and tilting or twisting of the stone with subsequent intermittent obstructions may cause exacerbation or renewal of the renal colic pain. The severity of the pain depends on the degree and site of the obstruction, not on the size of the stone. A patient can often point to the site of maximum tenderness, which is likely to be the site of the ureteral obstruction.

A stone moving down the ureter and causing only intermittent obstruction actually may be more painful than a stone that is motionless. A constant obstruction, even if high grade, allows for various autoregulatory mechanisms and reflexes, interstitial renal edema, and pyelolymphatic and pyelovenous backflow to help diminish the renal pelvic hydrostatic pressure, which gradually helps reduce the pain. The interstitial renal edema produced stretches the renal capsule, enlarges the kidney (ie, nephromegaly), and increases renal lymphatic drainage. (Increased capillary permeability facilitates this edema.) It may also reduce the radiographic density of the affected kidney's parenchyma when viewed on a noncontrast CT scan.

Distension of the renal pelvis initially stimulates ureteral hyperperistalsis, but this diminishes after 24 hours, as does renal blood flow. Peak hydrostatic renal pelvis pressure is attained within 2-5 hours after a complete obstruction. Within the first 90 minutes of a complete ureteral obstruction, afferent preglomerular arteriolar vasodilation occurs, which temporarily increases renal blood flow. Between 90 minutes and 5 hours after the obstruction, renal blood flow starts to decrease while intraureteral pressure continues to rise. By 5 hours after a complete obstruction, both renal blood flow and intraluminal ureteral pressure decrease on the affected side.

Renal blood flow decreases to approximately 50% of normal baseline levels after 72 hours, to 30% after 1 week, to 20% after 2 weeks, and to 12% after 8 weeks.

By this point, intraureteral pressures have returned to normal, but the proximal ureteral dilation remains and ureteral peristalsis is minimal.

Interstitial edema of the affected kidney actually enhances fluid reabsorption, which helps to increase the renal lymphatic drainage to establish a new, relatively stable, equilibrium. At the same time, renal blood flow increases in the contralateral kidney as renal function decreases in the obstructed unit.

In summary, by 24 hours after a complete ureteral obstruction, the renal pelvic hydrostatic pressure has dropped because of (1) a reduction in ureteral peristalsis; (2) decreased renal arterial vascular flow, which causes a corresponding drop in urine production on the affected side; and (3) interstitial renal edema, which leads to a marked increase in renal lymphatic drainage. Additionally, as the ureter proximal to the stone distends, some urine can sometimes flow around the obstruction, relieving the proximal hydrostatic pressure and establishing a stable, relatively painless equilibrium. These factors explain why severe renal colic pain typically lasts less than 24 hours in the absence of any infection or stone movement.

Experimental studies in animals have suggested that renal damage may begin within 24 hours of a complete obstruction and permanent kidney deterioration starts within 5-14 days. While some practitioners wait several months for a stone to pass in an asymptomatic patient, others argue that permanent damage is occurring as long as intervention is delayed. Based on personal experience and anecdotal cases, the author recommends waiting no longer than 4 weeks for a stone to pass spontaneously before considering intervention. Convincing asymptomatic patients of the need for surgical intervention may be difficult in the absence of a clear consensus in the urological community about the length of time to wait before surgical stone removal, fragmentation, or bypass.

If only a partial obstruction is present, the same changes occur, but to a lesser degree and over a longer period. Proximal ureteric and renal pelvic hydrostatic pressures tend to remain elevated longer, and ureteral peristalsis does not diminish as quickly. If the increased pressure is sufficient to establish a reasonable flow beyond the obstructing stone, glomerular filtration and renal blood flow approximates reference range baseline levels, although pain may be ongoing.

Phases of the acute renal colic attack

The actual pain attack tends to occur in somewhat predictable phases, with the pain reaching its peak in most patients within 2 hours of onset. The pain roughly follows the dermatomes of T-10 to S-4. The entire process typically lasts 3-18 hours. Renal colic has been described as having 3 clinical phases.

Acute, or onset, phase

The typical attack starts early in the morning or at night, waking the patient from sleep. When it begins during the day, patients most commonly describe the attack as starting slowly and insidiously. The pain is usually steady, increasingly severe, and continuous; some patients experience intermittent paroxysms of even more excruciating pain. The pain level may increase to maximum intensity in as little as 30 minutes after initial onset or more slowly, taking up to 6 hours or longer to peak. The typical patient reaches maximum pain 1-2 hours after the start of the renal colic attack.

Constant phase

Once the pain reaches maximum intensity, it tends to remain constant until it is either treated or allowed to diminish spontaneously. The period of sustained maximal pain is called the constant phase of the renal colic attack. This phase usually lasts 1-4 hours but can persist longer than 12 hours in some cases. Most patients arrive in the ED during this phase of the attack.

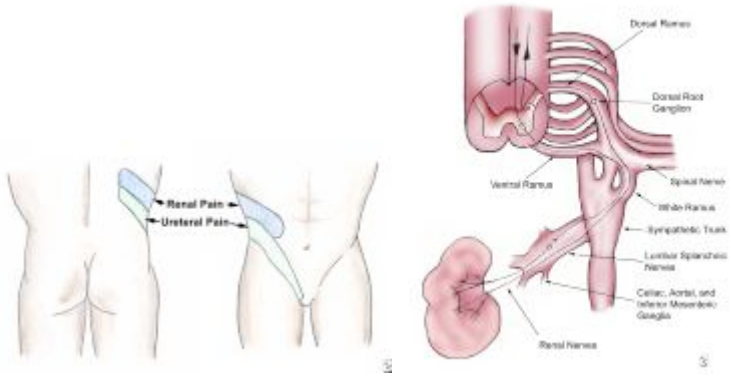
Abatement or relief phase

During this final phase, the pain diminishes fairly quickly, and patients finally feel relief. Relief can occur spontaneously at any time after the initial onset of the colic. Patients may fall asleep, especially if they have been administered strong analgesic medication. Upon awakening, the patient notices that the pain has disappeared. This final phase of the attack most commonly lasts 1.5-3 hours.

Pain in renal colic

Renal pain fibers are primarily preganglionic sympathetic nerves that reach spinal cord levels T-11 to L-2 through the dorsal nerve roots. Aortorenal, celiac, and inferior mesenteric ganglia are also involved. Spinal transmission of renal pain signals occurs primarily through the ascending spinothalamic tracts. In the lower ureter, pain signals are also distributed through the genitofemoral and ilioinguinal nerves. The nervi erigentes, which innervates the intramural ureter and bladder, is responsible for some of the bladder symptoms that often accompany an intramural ureteral calculus.

- Upper ureter and renal pelvis: Pain from upper ureteral stones tends to radiate to the flank and lumbar areas. On the right side, this can be



confused with cholecystitis or cholelithiasis; on the left, the differential diagnoses include acute pancreatitis, peptic ulcer disease, and gastritis.

- Middle ureter: Midureteral calculi cause pain that radiates anteriorly and caudally. This midureteral pain in particular can easily mimic appendicitis on the right or acute diverticulitis on the left.
- Distal ureter: Distal ureteral stones cause pain that tends to radiate into the groin or testicle in the male or labia majora in the female because the pain is referred from the ilioinguinal or genitofemoral nerves. If a stone is lodged in the intramural ureter, symptoms may appear similar to cystitis or urethritis. These symptoms include suprapubic pain, urinary frequency, urgency, dysuria, stranguria, pain at the tip of the penis, and sometimes various bowel symptoms, such as diarrhea and tenesmus. These symptoms can be confused with pelvic inflammatory disease, ovarian cyst rupture, or torsion and menstrual pain in women.

Most of the pain receptors of the upper urinary tract responsible for the perception of renal colic are located submucosally in the renal pelvis, calices, renal capsule, and upper ureter. Acute distention seems to be more important in the development of the pain of acute renal colic than spasm, local irritation, or ureteral hyperperistalsis. Stimulation of the peripelvic renal capsule causes flank pain, while stimulation of the renal pelvis and calices causes typical renal colic. Mucosal irritation can be sensed in the renal pelvis to some degree by chemoreceptors, but this irritation is thought to play only a minor role in the perception of renal or ureteral colic.

In the ureter, an increase in proximal peristalsis through activation of intrinsic ureteral pacemakers may contribute to the perception of pain. Muscle spasm, increased proximal peristalsis, local inflammation, irritation, and edema at the site of obstruction may contribute to the development of pain through chemoreceptor activation and stretching of submucosal free nerve endings.

Nausea and vomiting are often associated with acute renal colic and occur in at least 50% of patients. Nausea is caused by the common innervation pathway of the renal pelvis, stomach, and intestines through the celiac axis and vagal nerve afferents. This is often compounded by the effects of narcotic analgesics, which often induce nausea and vomiting through a direct effect on GI motility and through an indirect effect on the chemoreceptor trigger zone in the medulla oblongata. Nonsteroidal anti-inflammatory drugs (NSAIDs) can often cause gastric irritation and GI upset.

The presence of a renal or ureteral calculus is not a guarantee that the patient does not have some other, unrelated medical problem causing the GI symptoms.

Acute onset of severe flank pain radiating to the groin, gross or microscopic hematuria, nausea, and vomiting not associated with an acute abdomen are symptoms that most likely indicate renal colic caused by an acute ureteral or renal pelvic obstruction from a calculus. Renal colic pain rarely, if ever, occurs without obstruction.

In some cases, a stone may pass before the definitive imaging procedure has been completed. In these cases, residual inflammation and edema still may cause some transient or diminishing obstruction and pain even without any stone being positively identified.

Nerve blocks

Nerve blocks have been used successfully in both the diagnosis and treatment of renal colic, although they are more helpful in chronic than in acute cases. Intercostal nerve blocks can be used to differentiate pain from chondritis, neuromas, and radiculitis from true renal pain. This is achieved by injecting an anesthetic agent, such as lidocaine, around the 11th or 12th intercostal nerve proximal to the site of the pain at a time when the patient is experiencing pain. If the injection causes abolition of the pain, a peripheral nerve or musculoskeletal etiology is suggested. Subsequent injections of various agents to produce neurolysis (eg, 10% phenol or 100% absolute alcohol) have been tried but often result in an intolerable denervation-related discomfort.

In the mid 1950s, a study was reported in which 51 patients with documented renal colic were successfully treated for pain using only a unilateral posterior splanchnic nerve block. Complete relief from pain, nausea, and vomiting was reported in all 51 patients treated.

Urinalysis

Microscopic examination of the urine is a critical part of the evaluation of a patient thought to have renal colic. Gross or microscopic hematuria is only present in approximately 85% of cases. The lack of microscopic hematuria does not eliminate renal colic as a potential diagnosis. In addition to a dipstick evaluation, always perform a microscopic urinalysis in these patients.

Pay attention to the presence or absence of leukocytes, crystals, and bacteria and to the urinary pH. In general, if the number of WBCs in the urine is greater than 10 cells per high-power field or greater than the number of RBCs, suspect a urinary tract infection (UTI). Determining urinary pH also helps because, (1) with a pH lower than 6.0, a uric acid stone should be considered, and (2) with a pH greater than 8.0, an infection with a urea-splitting organism such as *Proteus, Pseudomonas,* or *Klebsiella* is likely. Urinary crystals of calcium oxalate, uric acid, or cystine may occasionally be found upon urinalysis. When present, these

crystals are very good clues to the underlying type and nature of any obstructing calculus.

While mild leukocytosis often accompanies a renal colic attack, a high index of suspicion for a possible infection should accompany any serum WBC count of 15,000/μL or higher in a patient presenting with an apparent acute kidney stone attack, even if afebrile.

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